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SCARLET FEVER AND RELATED STREPTOCOCCAL INFECTIONS*

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It is doubtful if a more controversial subject could have been chosen than that which I have been asked to discuss today. The general conception of scarlet fever and its relationship to other infections with beta hemolytic streptococci has changed considerably in recent years and indeed the disease scarlet fever itself has changed considerably within the past fifty years. Before discussing the more controversial aspects of these infections it might be well to review briefly their history.

Scarlet fever is the only streptococcal infection for which morbidity records are available and in so far as can be determined there has been no great change in its prevalence since records have been kept. There has, however, been a marked change in the severity of the disease. When first distinguished from other illnesses in the latter part of the 17th century, scarlet fever was considered a very mild disease. There have been, apparently, several distinct cycles in which the disease became severe, only to revert to the mild form again.

In the United States scarlet fever apparently reached a peak of severity during the last half of the 19th century and has steadily become less severe up to the present time. In those areas where comparisons can be made there has been a decrease in the case fatality rate of about 95% during the past 75 years. In other words, the ratio of cases to deaths has changed from approximately 5 to 1, to 120 to 1.

The decrease in mortality from scarlet fever, as shown by Ramsey[1] has not been uniform throughout the country. There has been a marked decrease in the mortality rates in the northern states but relatively little change in the southern states, at least during the past 20 years. Ramsey also showed that the decrease in mortality has been more marked in urban areas than in rural areas. There seems to be no obvious explanation of the differences in the trend in different parts of the country. It has been suggested that the more general use in the northern states of scarlet fever anti-toxin in the treatment of the disease and of the toxin as an immunizing agent might account for the differences that have been observed in these different areas. It should be noted, however, that the downward trend was well established before the development of either of these agents, and has not been materially accelerated since their introduction.

There is little evidence of change either in prevalence or in severity of the other manifestations of streptococcal infection. Wesselhoft[2] showed that according to the records of the Metropolitan Asylums Board Hospitals in London from 1897 to 1914, in spite of wide fluctuations in the incidence of scarlet fever, the incidence of otitis media remained almost constant. Mortality reports in general indicate no decrease in otitis media at all comparable to the decrease in mortality from scarlet fever. Puerperal sepsis, as measured by mortality records, decreased during the early part of the 20th century and then remained constant, or showed slight increases prior to the advent of sulphanilamide. It would be extremely hazardous to attempt a prediction of future trends in any of the streptococcal infections, but it certainly seems within the realm of possibility that scarlet fever may again assume a role of importance as a cause of death. For this reason, if for no other, continued efforts to develop means of prevention or amelioration seem important.

*Read before the Medical Society of Delaware, Rehoboth, September 10, 1940.

Any discussion of preventive measures or therapeutic measures immediately involves one in the controversial subject of the specificity of the etiological agent concerned in the various streptococcal infections. There is quite general agreement that scarlet fever is caused by streptococci capable of producing characteristic beta hemolysis on blood agar and that the organism falls into Lancefield's precipitin group A. The Dicks[3] in 1923 were able to produce scarlet fever in human volunteers by swabbing the throat with cultures of streptococci showing these characteristics and which were known to have been associated with clinical scarlet fever. The strains with which they were inoculated produced erythrogenic toxin which unquestionably was responsible for the toxic manifestations of the disease, particularly the rash. This ability of the organism to produce erythrogenic toxin has, however, been observed in many non-scarlatinal strains. It is also of interest that of the volunteers whose throats were swabbed with cultures known to be capable of producing typical scarlet fever, some developed angina with fever, indicating a successful invasion by the organism although they did not develop a rash or other evidence of the toxic effects of the infection. In view of subsequent observations, it seems probable that these individuals possessed at least some degree of anti-toxic immunity which was sufficient to prevent the development of the typical scarlet fever rash, but did not prevent invasion of the tissues by the organism.

Kirkbride and Wheeler[4] have shown that there is considerable variation in toxigenic activity of strains of streptococci isolated from scarlet fever as well as those isolated from other streptococcal infections. It would seem, therefore, that toxigenic activity cannot be considered as an indication of specificity of strains encountered in the different types of streptococcal infection.

Considerable progress has been made in the serological classification of the various strains of streptococci associated with human infections during recent years. By means of precipitin reactions Lancefield[5] was able to show that a large group of strains associated with human infection of various types had certain characteristics in common. This group she classified as group A as distinct from other groups largely non-pathogenic for man. She showed that by a relatively simple serological test the human pathogenic varieties could be broadly differentiated from the non-pathogenic. Griffith[6], more recently, was able by absorbed agglutinin technique to demonstrate some thirty distinct types, all but a small number of which also fell into Lancefield's group A. Some twenty of the Griffith serological types have been shown to be associated with typical scarlet fever, some much more frequently than others. These same types, however, have been found in tonsilitis, septic sore throat, erysipelas, otitis media, and other non-scarlatinal infections.

Clinical observations in common source outbreaks, in which it can be reasonably assumed that the infections were the result of exposure to a single strain of the infecting agent, appear to shed some light on the relationship of scarlatinal and non-scarlatinal strains of streptococci. During recent years in New York state a number of milk-borne epidemics of streptococcal infection have been closely studied. These epidemics have been classified either as outbreaks of scarlet fever or as epidemics of septic sore throat depending upon whether the typical scarlatinal rash was observed in any considerable proportion of cases. Approximately 1500 infected individuals were observed. The clinical manifestations in these cases, regardless of the general classification of the epidemic, were strikingly similar, with the exception of the presence or absence of the scarlatinal rash and desquammation. Without exception severe sore throat was a primary symptom. Fever was usually present and ranged from 100° F. to 104° F. The throat was almost invariably red and usually edematous. A punctuate rash was frequently seen on the palate, both in epidemics classified as scarlet fever and in those classified as septic sore throat. Inflammation of the tonsils was observed as frequently in epidemics of scarlet fever as in those classified as septic sore throat. In the epidemics classified as scarlet fever a typical scarlet fever rash was observed in approximately 60% of the cases, and as might be expected, the rash was observed more frequently in children than in adults. Eighty per cent

of the scarlet fever patients under 15 years of age developed the rash, as compared with 50% of those over 15 years of age. The rash was almost invariably followed by desquammation, but desquammation was rarely observed except in those individuals in whom an extensive rash had been observed. Those patients developing a scarlatinal rash were clinically indistinguishable from sporadic cases of scarlet fever or those observed in epidemics spread by contact. Cases in which no rash was observed occurring in epidemics classified as scarlet fever were clinically indistinguishable from cases occurring in epidemics of septic sore throat. The scarlet fever cases who did not develop a rash and the cases of septic sore throat were clinically indistinguishable from the type of cases frequently seen in the absence of any epidemic and usually diagnosed as severe tonsilitis or streptococcal tonsilitis.

An opportunity was afforded in the study of these epidemics to observe patients over a period of time and to observe the complications that developed in both types of infection. Approximately 25% of the patients seen in each epidemic, regardless of predominance of scarlatinal rash developed one or more serious complications. The complications most frequently observed were arthritis, rheumatism, otitis media, mastoiditis, quinsy, cervical abscess, nephritis, pneumonia, sinusitis and erysipelas. A significant portion of the cases in each study did develop these other types of infection which have been claimed to be specific types of infection and the organisms having a definite specificity. The same complications were observed in all of the epidemics and each of the complications occurred with approximately the same frequency whether associated with a scarlatinal rash or not. There were 16 deaths among the 806 cases of scarlet fever and 8 deaths among the 723 cases of septic sore throat. Complications were given as contributory causes of death in 12 of the 16 deaths occuring in cases in which the disease was clinically classified as scarlet fever. Of the 8 deaths in individuals suffering from septic sore throat all were in individuals who developed complications of the original infection.

It has been assumed that an attack of scarlet fever produces immunity to subsequent infection with the same or similar strains of the infecting agent. Repeated typical attacks of scarlet fever are relatively rare. Observations in common source outbreaks of streptococcal infection, however, seem to indicate that this immunity is more apparent than real. In a number of common source outbreaks it has been apparent that a previous attack of streptococcal infection diagnosed as scarlet fever produced little or no immunity to subsequent infection. However, the subsequent infection did not as a rule result in the production of a rash even though the infection was with a good toxin-producing strain, indicating that there was immunity only to the erythrotoxin or rash producing factor.

It is generally agreed that an attack of scarlet fever usually results in reduced skin sensitivity to erythrogenic toxin and the Dick test has been demonstrated to be a reasonably adequate measure of anti-toxic immunity. It has also been shown that skin sensitivity to so-called scarlatinal toxin decreases with age even in the absence of known history of clinical scarlet fever, indicating the production of anti-toxic immunity through sub-clinical infection with erythrogenic toxin producing organisms.

We have been able to skin test considerable numbers of individuals having suffered from streptococcal infection in common source outbreaks classified either as scarlet fever or septic sore throat. It has been found that following infection with strains of an organism, infection with which results in a high proportion of cases characterized by a typical rash, there is a marked decrease of skin sensitivity.

In those individuals suffering from streptococcal infections in which rash was not a frequent observation very little change in the skin sensitivity has been observed following the illness. This would seem to indicate considerable difference in toxin-producing properties of the different strains involved, whether they are strains associated with clinical scarlet fever or with the other types of infection.

Another evidence of anti-toxic immunity

derived from previous experience with the toxin-producing strains of streptococci is the close parallel between the age distribution of skin senstivity as measured by the Dick test and the age distribution of scarlet fever rash in epidemics of scarlet fever. Approximately 7500 individuals were skin tested with a toxin of the New York 5 strain in communities in New York state in which the incidence of scarlet fever was not and had not recently been high. It was found that the percentage of positive skin reactors decreased consistently with increasing age and that this decrease in skin sensitivity was closely paralleled by the decrease in the appearance of the typical scarlatinal rash in epidemics of scarlet fever. This observation would seem to be additional evidence that the appearance of the scarlatinal rash in infections with organisms capable of producing scarlet fever depends upon the individual's anti-toxic response to previous infection.

CONCLUSIONS

Clinical, bacteriological, immunological and epidemiological evidence all seem to indicate that scarlet fever is not a distinct entity but that at the present time at least, it is one of the less important manifestations of infection with the general group of streptococci associated with a large group of disease processes. It seems most probable that host reaction to infection depends upon a number of factors. Important among these factors are:

1. Characteristics of the particular strain of the infecting agent.
2. The portal of entry of infection.
3. Previous streptococcus experience of the host.

If this conception of scarlet fever and other streptococcal infections is to be accepted, and the evidence does not, in my opinion, justify any other conception, present methods of prevention and treatment need radical revision. Isolation of scarlet fever is no more important than isolation of tonsilitis, pharyngitis, or possibly otitis media of streptococcal origin. If a satisfactory artificial immunity is to be produced an anti-bacterial as well as an anti-toxic immunity must be the ultimate aim, and in view of the multiplicity of strains involved any antigen used for immunization must have a broad valence. It should also be borne in mind that biological therapy, to be of general usefulness, must combine anti-bacterial with anti-toxic properties and be either type specific or of multiple valence.

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State Board of Health

DISCUSSION

PRESIDENT BARNES: Thank you, Dr. Stebbins.

At this time I would like to ask Dr. Cameron to discuss Dr. Stebbins' paper. I will present Dr. Cameron to the Society as a newly appointed Executive Secretary of the State Board of Health. Dr. Cameron.

DR. EDWIN CAMERON (Dover): Because of the lateness of the hour I would like to confine my discussion solely to complimenting Dr. Stebbins on his paper.

I think that we can with a reasonable amount of assurance look forward to a possibility of the return of a much more virulent strain of scarlet fever organism than we now encounter. We always have been aware of the gradual and very marked decline in the virulence of the attacks of scarlet fever.

From a practical public health point of view, while scarlet fever itself does not account for a high mortality among the people who have it, at the same time it is one of our most perplexing problems in regard to the control of the disease. Diagnosis is not always clear, and in addition to that, of course, we have school authorities to contend with who dislike very, very much our diagnosing and attempting to control scarlet fever through isolation.

I think I will conclude by thanking Dr. Stebbins; and again let me compliment you on your paper.

DR. G. J. BOINES (Wilmington): I would like to ask Dr. Stebbins what importance he attaches to a Schultz test and whether he would isolate a septic sore throat case as much as he would the scarlet fever case; and whether there is any method he uses to differ-

entiate between a septic sore throat with a very mild rash and a scarlet fever case without a rash—would he attempt any differentiation?

DR. STEBBINS: As to the question regarding the Schultz-Charlton reaction, that is obviously, I believe, merely a reaction of toxin with antitoxin, and if you inject antitoxin it neutralizes the toxin and you get a blanching of the rash. I don't think that in itself it is a specificity of the organisms or of the toxin. The antitoxin used in the test is almost always of a multivalency. We in New York state use an antitoxin produced of the New York 5 strain which we know is effective in neutralizing the toxin of at least 15 different Griffith serological strains. So it is not surprising that the injection of the antitoxin into the skin will blanch the rash produced by the toxin.

As to the differentiation of mild scarlet fever and septic sore throat, the cases that we have seen have been entirely comparable in every respect, except the presence or absence of the rash. The complications are of the same nature, and of the same frequency. In the group that we have studied there has been a considerably higher case fatality rate in scarlet fever than in septic sore throat. But that difference is not of statistical significance, I believe. The fatality rate in both types of infection where the cases have been followed over a period of time and we know of the development of other streptococcal complications has been many times higher than the fatality rate in sporadic cases, or reported cases, where there has not been that continued followup. We believe that is merely an expression of the missed diagnoses, that is, a patient has scarlet fever and is reported as scarlet fever and develops some obviously related complication; and yet there is sufficient time following the original attack so it is not attributable to the scarlet fever.

We think the fatality rate that has been observed in these epidemics, while it may be influenced by the fact that in a common source outbreak frequently the patient gets a much larger slug of the infecting material, is higher than the figures would indicate.

As to the question of the isolation of the other manifestations of the streptococcal infection, I do not believe we can justify any difference in the isolation precautions for any of these. It is obvious that you can contract scarlet fever from contact with a case of otitis media. It has happened repeatedly. Gordon in Detroit has shown through a long experience that a large proportion of the scarlet fever came into a hospital there from people contracting that infection. In people not having scarlet fever they have discovered sources of infection. In the average case of scarlet fever less than one in ten can be shown to have contracted it through direct contact with scarlet fever. Carriers undoubtedly play an important role, but the carriers of all of these different strains of organisms apparently have approximately the same ability to produce scarlet fever as any one of the others, and we have seen typical scarlet fever contracted from what has been diagnosed as septic sore throat, just streptococcal tonsilitis, or severe tonsilitis.

OTITIS MEDIA

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Otitis media is frequently seen by the general practitioner as well as by the otologist. Prompt recognition and early treatment produce quick relief of pain, shortening of the period of disability, and prevention of sequellae. Of all the pains we mortals are given to earache must be near the top of the list in intensity. The old methods of allowing the eardrum to rupture spontaneously or with the help of tobacco juice, leeches, etcetera, have given place to more modern and effective methods. Days and weeks of lingering illness, fever, and frequent nights of pain are now prevented by early treatment. Statistics of morbidity, mortality, and complications have been radically revised in the last few months. Sulphanilamide and sulphapyridine have drastically reduced the number of operations for mastoiditis which formerly followed otitis media. The profession awaits with interest the place other new sulpha drugs will take in treating otitis media.

The first type of otitis media I wish to consider is one which gets less than its deserved amount of attention:

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ACUTE SEROUS CATARRH OF THE MIDDLE EAR

It is the most common disease of the middle ear yet one which is seldom recognized. This condition usually follows inflammation of the eustachian tube which closes, or may be due to pharyngeal stricture of the eustachian tube. At any rate the air of the middle ear is resorbed, and the drum is retracted. If there is a negative pressure in the middle ear, mucus is aspirated from glands of the eustachian tube, or a transudate from the blood vessels of eustachian tube.

Symptoms are very significant:

1. Sudden onset of deafness.
2. Patient complains that his own voice bothers him, makes him nervous to talk, and that his voice makes an echo.
3. Dependence of hearing on position of head—if fluid goes away from window the hearing is better.
4. Feeling of fullness in ear.
5. Itching in ear.
6. Pain around ear, especially in the sulcus between the mandible and sternocleidomastoid muscle.
7. Mild dizziness and tinnitus.

The ear drum is usually retracted, glistening, and yellowish or brownish in color. The fluid may not fill the middle ear, when the lower part may be yellowish and the upper part bluish. If the middle ear is not full there may be a level line which changes with movement of the head. If the cellar is very deep, fluid may not show because it may not reach the drum. In such a case only a retracted glistening drum is seen. Acute serous catarrh of middle ear frequently recurs and adhesions form which divide the middle ear into spaces. In this case there may be more than one cone of light.

Treatment—Air inflation without catheter by the Politzer method will remove the fluid and restore the hearing, if fluid is serous. If mucus is present, then it is necessary frequently to do a myringotomy, and blow out the fluid by the Politzer method. It is very important to pay careful attention to the nasopharynx. Adenoid hypertrophies are to be removed, especially if there are encroachments on the openings of the eustachian tubes.

ACUTE OTITIS MEDIA

This condition is frequently seen in infants, children and adults. It is often bilateral in infants because of the shortness and large caliber of the eustachian tubes. Otitis is a pure infection caused by streptococci, influenza bacilli, diplococci, and pneumococci. An uncontaminated culture may be taken through a micropipette drawn to a very small point, and introduced through the unbroken ear drum after sterilizing the external ear canal by the use of tincture of metaphen or some similar antispetic. If the pipette with a rubber bulb is not handy, then the blade of the knife which is used for myringotomy may be used to inoculate a tube of liquid media which is then incubated in the usual way. A culture taken from the ear canal after myringotomy or when the ear is discharging is of very little value because of contamination. Staphylococci alone usually are not found in the middle ear cavity.

The uncomplicated streptococcic otitis usually starts with pain which is sticking in character but not severe at first. Hearing is diminished slightly and tinnitus is present but not marked. There is slight dizziness, but fever may be absent in the early stages. The eardrum is red over the malleus at first. In a few hours the whole drum becomes red and is covered with white spots. Landmarks of the drum become indistinct and finally obliterated. The drum then is bulging and macerated. There is now a yellowish serous exudate behind the ear drum indicating a collection of pus. By this time the pain is severe and fever may be high. Treatment in the first stage frequently will cause a subsidence of the condition. Phenol, 2%, and glycerine, 98% is warmed and dropped in the ear canal in doses of two or three drops every two or three hours. Dry heat by infra-red lamp, electric pad, or hot water bottle is very comforting to the patient. Heat is so appreciated that the patient frequently burns the face or external ear, not realizing how high the temperature becomes. The patient should be in bed and should have general treatment for the cold which usually accompanies otitis media.

Sulphanilamide is given in doses of ten grains every three or four hours for adults until definite improvement occurs or until the patient shows toxic effects from the drug.

The dose is then reduced according to the indications mentioned above.

Not to be forgotten is the attention to the nasopharynx. Ephedrine sulphate 1% in normal salt solution should be dropped in the nostrils with the patient in the lying position, having the head dropped over the side of the bed to insure the solution's reaching the desired places. After waiting a few minutes an antiseptic solution such a argyrol, collargol, or neosilvol, is then dropped in the nostrils to act on the nasopharynx and openings of eustachian tubes. After a few hours of the above treatment the patient is relieved of pain (the fever has subsided) or the patient is worse and the ear drum is bulging. At this point the ear canal should be sterilized and under a general anaesthetic such as nitrous oxide, the ear drum is incised. The incision is made in the bulging portion of the drum which is usually the inferior and outer quadrant. In infants, the drum is incised without any anaesthetic. If a general anaesthetic is contraindicated in an adult then a mixture of cocaine crystals, phenol and menthol in equal parts may be applied to the drum 10 minutes before the incision is made. This method has the objection of macerating the ear drum. After myringotomy the pain subsides in about one half hour and drainage becomes established. If the myringotomy was done early enough the discharge may be clear and of short duration. If myringotomy was delayed the drainage is thicker and continues longer. Drainage may last from a few hours to two or three weeks. As a general rule if an acute otitis media has not stopped discharging in three weeks a mastoidectomy will be necessary. After the myringotomy general treatment is continued as outlined above. Many patients remain ambulatory but quicker recovery follows if the patient is in bed. The external canal may be irrigated with warm boric acid solution every three hours or often enough to prevent purulent material accumulating and obstructing the opening in the ear drum. Some men prefer a gauze wick to absorb the drainage instead of using irrigations.

The external meatus should be touched daily with a 5% solution of silver nitrate to prevent the development of external otitis. When the discharge becomes scanty after a few days, local treatments with a cold quartz applicator seems to be beneficial.

While the ear is discharging it is important to continue attention to the nasopharynx as mentioned above. When the incision is healed an air inflation by the Politzer method is effective in clearing the middle ear cavity and restoring the hearing.

After the myringotomy has been done and the culture is reported, then it is necessary to decide whether or not to continue the administration of sulfanilamide. If a streptococcus hemolyticus is reported then the sulfanilamide should be continued. If pneumococcus is present sulfapyridine should be used instead. In my experience and in the experience of numerous writers of articles published in recent medical literature, sulfanilamide has been given great praise in shortening the course and preventing complications in otitis media.

Babcock of New York, in the August, 1940, issue of Archives of Otolaryngology, questions the value of sulfanilamide by presenting a series of 103 cases about one half treated with sulfanilamide and the other half treated without. Most of the literature is overwhelmingly in favor of the use of sulfanilamide and shows that complications are greatly reduced by its use. It is probably a little bit too early to decide entirely about the value of it because it is known that the complications vary from year to year and in different localities. We seem to have fewer complications and fewer cases of mastoiditis in this section than they have in Philadelphia, or than they have in Wilmington, and New York apparently has more than they have at Philadelphia.

In closing I wish to emphasize three points:

1. The importance of early diagnosis.
2. Myringotomy promptly after bulging of the drum appears.
3. Sulfanilamide or sulfapyridine as part of the treatment.
4. Attention to the posterior nasopharynx.

DISCUSSION

DR. I. W. MAYERBERG (Dover): Dr. Marvil has presented this paper on acute otitis in such a thorough manner that it leaves very little room for discussion. His classifications

have been fine. In that serous or tubo type of acute otitis media, during the course of an acute infection, a doctor will call in an otologist sometimes because he is on his tiptoes, and knows that an otitis media will probably develop. He looks in there and sees the very red, fiery-looking drum. He doesn't feel as if there is any bulging there, the child doesn't complain of very much pain, but still the ear drum looks so congested, and it looks as if there is so much trouble in that ear that he will call upon the otologist to do something about it. I have found that these doctors seem a little bit disappointed sometimes if the otologist doesn't recommend a myringotomy.

I thoroughly agree with Dr. Marvil that myringotomy in that stage of the serous type of otitis does not need drainage. It will resolve mainly on its own accord, as he says, inflation carefully done is the thing to do in that case. After all, it is just a mechanical process. I try to picture that process in my mind as an automatic valve cutoff, and carried a little bit further it looks to me just like a floating ball in a toilet tank; as it reaches its level it will stop by itself. In other words, by closing the eustachian tube we have a negative pressure in the ear drum, we have transudation, but there won't be any more exudate formed in that ear until you strike a level, and then the eustachian tube will open in a majority of the cases, and that is drained off or absorbed.

Otitis media should be looked at as something that is always dangerous. The complications, if untreated, are sometimes very serious, and we should bend all our efforts to treatment. I believe, with the doctor, that we should use some sterilization in the shrinking process of the nasopharynx. I believe if sulfanilamide is indicated that we should institute that method. Sometimes adenoids in the postnatal space itself are not a causative factor because they don't press upon the eustachian orifices. They make pressure on the veli palatini muscle that keeps the eustachian tube from opening as it should properly.

I don't know about the use of sulfanilamide. I am using it, and I am wondering if it is in the scope of this discussion of the acute condition for us to discuss a chronic condition. Should we continue to use the sulfanilamide and the sulfapyridine in those cases produced by the pneumococci, and if we should continue the process without surgical interference should we, as is our usual custom, keep tab by clinical symptoms and laboratory methods, or not? I am fond of Schilling's differential white count as a guide.

DR. A. J. STRIKOL (Wilmington): I have very little to say. In fact, I didn't expect to get up and discuss the paper. It was well presented.

Possibly I could add one more thing to it. Dr. Crowe of Johns Hopkins and his associates are doing something that we haven't been doing in Wilmington or in most of our cities. They are treating the nasopharynx, especially the lymphoid tissue around the orifice of the eustachian tube, which completely or partially prevents drainage as well as aeration of the middle ear. If that isn't done, quite often the child keeps getting worse, and a certain amount of impairment is produced, possibly a permanent impairment. Dr. Crowe has wonderful results. We have sent some of our cases down there and put them in their hands, and have had remarkable results.

As far as the other treatment is concerned, sometimes I think even the ear men are doing more harm than good by putting various solutions in the ear. When you have a small perforation the size of a pinhead—and that cavity is a pretty good size in the middle ear—filled with mucopurulent secretion, and then put in some antiseptic like metaphen or alcoholic glycerin, I don't know how much good it does. I doubt very much whether it does any. In fact, possibly it produces more irritation, sets up an inflammation, and prolongs the condition.

As far as the sulfanilamide is concerned, of course there are two schools at the present time. In my own hands in some cases the results with sulfanilamide were excellent. I think the crux of the thing lies in early recognition and treating the nasopharynx. In children, as the doctor said, the eustachian tube is short and of larger caliber, just like a tunnel, and the infection travels from the nasopharynx into the ear. That is why the more purulent it is the more liable it is to go into the middle ear. I think we ought to do

a little more cleaning out of the nasopharynx, not by putting various strong solutions in the nostrils but by gentle suction, capillary suction. Some of us know how adults blow their noses and get rid of that stuff, but children lie in bed with their noses filled and the nasopharynx completely blocked with mucopurulent secretion, and very little help is really given. I think the sooner we start to clean out that nasopharynx and treat the upper respiratory tract, the better. Nature takes care of the rest pretty well.

I don't think our antiseptics are worth very much, except sulfanilamide or sulfapyridine. I think they do some good.

As far as the mastoids are concerned, we don't do as many mastoids, not only because of the sulfanilamide, but for other reasons. Most of the general men have otoscopes. A few years ago there were very few who had otoscopes and very few men looked in the ears. A child was treated for some other condition. Now they certainly are on their toes, and on our heels, especially the pediatricians. That is almost the first thing they do. They go into the pediatric wards, and I think they spend a great per cent of the time looking in the ears, and rightly. I think we are preventing complications where they occurred previously. Because of the eustachian tube being completely clogged, the infection goes back into the mastoid cells and because of improper drainage sets up an inflammation, and so forth—just a vicious circle, and we have to open it up in back to establish drainage.

In children we did not do a complete mastoidectomy, we do an antrotomy, really, remove the cortex, make a hole in there, and gave nature a chance to drain it out. After all, we still have to depend on nature, not so much on drugs. Give nature the chance.

Another thing: the children lie in bed right on their backs without changing their position, and of course the secretion is in the most dependent part, in the nasopharynx around the orifice of the eustachian tube. I think the child's position should be changed. I think that sometimes we keep the room too warm, too dry, with poor ventilation. I think a little exercise is a help, if not out of bed, in bed; in other words, stimulate the ciliary action of the nasopharynx in getting the secretion out of there. If you get it out of the nasopharynx you will have less trouble.

I enjoyed the paper and the doctor certainly has covered it very thoroughly. I think we are improving in our method of treatment. Thank you!

DR. E. R. MAYERBERG (Wilmington): I thoroughly enjoyed Dr. Marvil's paper and the special thought of his paper. There are two or three things I would like to discuss, though. The first is as to Dr. Marvil's suggestion as to the treatment in the early cases of the serous otitis media. My feeling is that every serous condition has an underlying inflammatory basis, and when you have an underlying inflammatory basis you usually have bacteria present somewhere. For that reason I feel that inflation of the eustachian tubes by any method, particularly the Politzer method, is bad practice.

In the first place, the Politzer method of inflation is a hit-or-miss method. It is almost impossible to get a child or young person to cooperate in the performance of that particular method. You all know what it is. Just for the moment I will briefly review it. It depends upon cooperation and coordination. You give the patient a sip of water and ask the patient to swallow while you blow air through the nose, with either a compressed air outfit or a Politzer bag. The average adult can do that, the average child or young person cannot. In the first place, they are scared to death when you start your treatment. In the second place, when the air comes they open their mouth and spit water all over.

The second reason why it is not successful and should not be used is because nobody in the world can make that air go through one eustachian tube. If one ear is involved you certainly do not want to inflate both ears. Usually when the Politzer method is successful both tubes are inflated. The danger is, if there are organisms in the nasopharynx they may easily be blown through the eustachian tube into the other middle ear, and instead of having one otitis media you have a bilateral one. If the inflation method is to be used the Valsalva is the best method.

Now as to treatment, I am so glad to hear Dr. Marvil supplement my own theory.

Paracentesis means a puncture of any organ. A paracentesis of the ear drum means that you puncture the ear drum, while a myringotomy means that you incise the drum. Myringotomy should be done under general anesthetic, particularly in young children. You cannot rely upon them to sit still or stand still while you do it. The slightest movement will misdirect your knife and great harm can be done to the ear structures, so a general anesthetic like nitrous oxide or ether should be given, nitrous oxide for children over six and ether for those under six.

The after treatment: it has been my feeling for years that more mastoids have been caused by the irrigation of the acute ears than anything else. It is usually done by inexperienced hands, the solutions are carried into the canal under pressure, and the infected material is carried through the middle ear back into the mastoid cyst. A myringotomy gives you drainage and all that is necessary is to keep the canal clear. That is best done by using applicators with the long tapers of cotton to act as sponges, carrying that down in the canal, several of them, one after the other, and most of that heavy purulent material can be removed by that method. Any drop that you want to use—it is immaterial which one you use—can be applied, cotton kept in the ear, and can be changed frequently during the day.

Cleanliness is our watchword in the treatment of these cases. Whoever has charge of the individual should be instructed to wash her hands first always, use alcohol on them, and keep the cotton under sterile conditions, not to do as one trained nurse did in a case of mine that had scarlet fever. The child had an ear drum opened because of an acute otitis. I went in there one morning and found the mother crying. I asked what was the matter.

"Well," she said, "I went into the child's quarters and found the nurse actually using some cotton out of an old teddy bear that had been lying around on the floor two or three years." She was using that in his ear. The mother always blamed the nurse because the child had to have a mastoidectomy a few days after that. So cleanliness is the watchword.

As far as the incidence of mastoiditis goes there has been a noticeable decrease in the last two or three years. Whether it is because the pediatricians and the general physicians are making early diagnoses and are instituting treatment themselves or having someone else do it, I do not know. My feeling that the sulfanilamide is largely responsible for it. Instead of doing fourteen or fifteen mastoids in a season, last year I did four or five. And I want to say this: that every single one of those cases were from outside of Wilmington. That is one reason I say that I think sulfanilamide and the allied preparations had something to do with it, because the men in Wilmington are using these preparations. Men in Maryland, Pennsylvania and New Jersey, where most of these cases came from, had not used any treatment.

I know that at one of the Academy meetings last year Dr. Hauser of Philadelphia reported that in his group of cases sulfanilamide had shown very little effect, but I believe, as Dr. Marvil says, the individuals will show a different picture, that the administration of these drugs early and the proper treatment of otitis media, surgically if necessary, will reduce the incidence of mastoiditis. Thank you!

DR. MARVIL: I think the discussion was very much in order. About the inflation: in my experience I have never seen a case where there was a spread of infection from one side to the other by the inflation method. It is true it is a hit-or-miss method, but all you have to do is to put a speculum in and look in the ear again, and you can tell whether your first squeeze of air did the work, if you haven't already had a otoscope or a tube in the child's or the adult's ear. If the first one didn't work, you can use the second one; and as far as that goes, you usually don't have to do anything, because the child says, "I can hear," as soon as you get the middle ear clear.

The other things I think were very much in order and I appreciate them very much. I think they brought out points that I didn't bring out, but since most of our men here do general practice instead of specialized practice I didn't go into the details so very much because I didn't think it would be of interest to most of the men here. Thank you!

EDITORIAL

DELAWARE STATE MEDICAL JOURNAL

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VOL. XIII MARCH, 1941 No. 3

RE: LIFE INSURANCE

The question of physicians' life insurance in time of war is one of vital importance to the practitioner and his family. The discussion in the state medical journals began in the *New York State Journal of Medicine* for July 15, 1940. The October issue of the *Connecticut State Medical Journal* made reference to this New York editorial, to which the New York editor referred (November 1, 1940) as follows:

Our neighbor chides us editorially—in kindly fashion—for having ventured, in our military medicine issue, to raise some questions concerning the effect of peacetime mobilization on physicians' insurance policies and programs. "*The New York State Journal of Medicine*, in an editorial entitled 'Protecting Insurance in Wartime,' makes a venturesome expedition into a field in which more authoritative opinion may be obtained here in Connecticut." One gathers the impression that the *Connecticut State Medical Journal* feels that we of New York have been carrying coals to Newcastle in the matter of insurance, that the voice of experience and authority should emanate from Hartford, and that —in good time—it will.

Continuing the subject, the New York editorial for February 15, 1941, says:

Well, *in loco parentis, the Connecticut State Medical Journal* spanks us again editorially in its January, 1941, issue, to wit:

"*The New York State Journal of Medicine* has recently given prominence to the discussion of the physician's life insurance in war time. In order to intercept any false interpretations or deductions a clarification of the subject is imperative. The voice of experience and, may we add, of authority, which the New York editor is pleased to ascribe to us, should logically come from Hartford, the insurance center of the nation. The payment of all types of insurance premiums becomes a serious problem for either physician or layman in the event of military service. Insurance carriers of accident and health policies are ruling that such policies shall continue in effect only provided that the insured remains within the United States or Canada, that he does not enter the air service, and provided that this nation does not engage in actual warfare. Life insurance is, of course, only one of the serious financial problems facing physician or layman who enters military service. He may be encumbered with mortgage payments on his home, monthly installments on his automobile or gas refrigerator, or interest on financial loans. The pay of a first lieutenant or of a captain will undoubtedly fall far short of the necessary income to meet these obligations.

"Our friends in New York commit a fundamental error when they reason that to borrow on a life insurance policy represents the insured's own money loaned back to him by the insurance company. A man does not borrow 'his own' money when he borrows on a policy, but, rather he enters into an agreement with the investment department of the insurance company whereby this same company agrees to invest some of its money with him, provided he pays interest for the use of it. This process is entirely comparable to the investment of money by the life insurance company in real estate and in investment securities, but in any event the company must realize a return on money invested in order to maintain solvency. Borrowing money from an insurance company by an individual is no different from borrowing money from a bank. In the second instance the bank requires collateral of securities, or other tangibles, while the insurance company accepts a life insurance policy as collateral up to the amount of reserve which has accrued against the policy.

"Automatic extension of insurance at the face value of the policy for a period dependent upon the amount of cash reserve in the policy has been suggested as a solution of the physician's difficulties in meeting premium payments. The question is, why limit the moratorium to life and accident premiums? It could as fairly be ap-

plied to any current debt or obligation incurred by any individual who may become a part of the fighting forces of the United States. What would be the result upon the economic system if such a procedure became legalized? Let us not forget that a life insurance policy may be paid for in full if the insured so desires. Furthermore, if a continuation of the payment of life insurance premiums is not desired for any policy, the insured is protected through paid-up insurance for 100% value of all premiums paid up to the time of the cessation of premium payments and, therefore, no further premium payments need be paid.

"Our neighbors from New York, 'to provoke discussion of this subject,' have suggested a transfer of insurance from private to government control. This invites the entrance of the government into the field of insurance. During World War I the government sold cigarettes at seven cents a package at army posts, while the same product was sold elsewhere at fifteen cents a package. How was the difference in selling price made up? How will the difference between the premium charged for government insurance and the actual cost of the insurance to the government be met? The answer, obviously, is by taxation toward which everyone contributes. How far do we wish to permit the government to be projected into private enterprise? If it is going into business at all, why not into all fields? Can the government handle any business as efficiently as is possible under good private management? How great a degree of efficiency in business may one expect from the government when the results of lack of efficiency, lack of judgment, lack of foresightedness, and lack of honest endeavor can always be compensated for by an increase in taxes? Will the change of operating personnel under government control due to the change in administrations tend to keep the level of efficiency as high as under private enterprise?

"It requires no deep study on the part of well informed citizens to realize the repercussions of the T. V. A. experiment where taxation is concerned. Private enterprise is based on the premise, 'be successful or you perish,' and that is an impelling incentive for the achievement of success. Such an incentive would be non-existent under government control. Government securities do not in themselves yield sufficient income to insure the solvency of any insurance organization. The government itself can make up this deficiency only by increased taxation, but the source of supply will fail if business is choked by a moratorium and the natural flow of money necessary to carry on all business is thereby reduced.

"There is most assuredly no novel or magic way of providing for life insurance premiums of those engaged in the business of military training. The mysteries of the Aladdin-like legerdemain performances in Washington have long since been divulged. Financial obligations, including life insurance premiums, cannot be suspended nor can protection exist for the physician only insofar as he pays for it. The hard way remains the only way for us, as for countless others in the disrupted world of today."

Delaware enters the discussion to ask, with all due humility, just one question: Is it not possible that our Connecticut confrere is in error, in Paragraph Two above? He infers that the premiums paid represent insurance only. It is common knowledge that these premiums are "loaded," and *do* represent savings as well as insurance! If you want to know what insurance only costs, get the rate for term insurance—there is practically no loading here and the savings item is entirely eliminated. Then compare this rate with that for ordinary level premium life insurance—you'll be surprised! In fact, the former, during the earlier age periods, is just about half the latter.

So, when a policy holder borrows on his policy he literally *is* borrowing his own money—part of the loan represents savings that the insurance company has wrung from him in addition to the cost of insurance alone.

We do not live in "the insurance center of the nation," but we are not at all convinced that "our friends in New York commit a fundamental error." We do not desire to have the New Deal take over the insurance business—God forbid; but the impending investigation of the business may very well look into this matter of premium rates, to the considerable profit of some 64,000,000 policy holders.

The Directory Page in this issue shows three changes from that published in the January issue. These are in the Special Committees, appointed by the President, and are made to avoid duplication of personnel.

The county and other societies are urged to send in their revisions immediately following their elections.

1941 John Phillips Memorial Award

On the recommendation of the Committee on Fellowships and Awards, the Board of Regents of the American College of Physicians, by unanimous resolution, has voted that the John Phillips Memorial Medal for 1941 be awarded to Dr. William Christopher Stadie, Associate Professor of Research Medicine at the University of Pennsylvania, Philadelphia, for his significant contributions to the knowledge of anoxia, cyanosis and the physical chemistry of hemoglobin, and more especially for his recent studies on the subject of fat metabolism in diabetes mellitus.

1789—MEDICAL SOCIETY OF DELAWARE—1941

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Meets Third Tuesday

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Alternates: C. M. Moyer, E. L. Stambaugh, J. R. Elliott, F. I. Hudson.

Censors: H. E. LeCates, Bruce Barnes, A. C. Smoot.

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DELAWARE STATE DENTAL SOCIETY—1941

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DELAWARE ACADEMY OF MEDICINE

Mrs. Ava Taylor Watson became librarian to the Delaware Academy of Medicine on September 1, 1933, and held this position to the day of her death, February 14, 1941. The success of the Academy has to a large measure been due to Mrs. Watson's untiring, whole-hearted, painstaking interest in the development of its library, and this stands as a monument to her memory.

Mrs. Watson was born in East Market, a town in Dorchester County on the Eastern Shore of Maryland, on January 11, 1891. She received her A. B. degree from Western Maryland College, Westminster, Maryland, in 1911. She received her degree in Library Science from Drexel Institute, Philadelphia, in 1931.

Before she undertook the organization of the library at the Delaware Academy, Mrs. Watson was associated with the Wilmington Public Library for a short time, and then with the Public Library in Charlottesville, Virginia. She was also connected with the library of the University of Florida.

The unexpected and untimely death of Mrs. Watson is a great personal loss to her many friends, and an incalculable professional loss to the Academy.

"The Foundation Prize" of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons

(1) "The award which shall be known as 'The Foundation Prize' shall consist of $150.00."

(2) "Eligible contestants shall include only (a) interns, residents, or graduate students in Obstetrics, Gynecology or Abdominal Surgery, and (b) physicians (with an M. D. degree) who are actively practicing or teaching Obstetrics, Gynecology or Abdominal surgery."

(3) "Manuscripts must be presented under a nom-de-plume, which shall in no way indicate the author's identity, to the Secretary of the Association together with a sealed envelope bearing the nom-de-plume and containing a card showing the name and address of the contestant."

(4) "Manuscripts must be limited to 5000 words, and must be typewritten in double-spacing on one side of the sheet. Ample margins should be provided. Illustrations should be limited to such as are required for a clear exposition of the thesis."

(5) "The successful thesis shall become the property of the Association, but this provision shall in no way interfere with publication of the communication in the Journal of the Author's choice. Unsuccessful contributions will be returned promptly to their authors."

(6) "Three copies of all manuscripts and illustrations entered in a given year must be in the hands of the Secretary before June 1st."

(7) "The award will be made at the Annual Meetings of the Association, at which time the successful contestant must appear in person to present his contribution as a part of the regular scientific program, in conformity with the rules of the Association. The successful contestant must meet all expenses incident to this presentation."

(8) "The President of the Association shall annually appoint a Committee on Award, which, under its own regulations shall determine the successful contestant and shall inform the Secretary of his name and address at least two weeks before the annual meeting."

JAS. R. BLOSS, M. D., Secretary
418 Eleventh St., Huntington, W. Va.

BOOK REVIEWS

Introduction to Dermatology. By Richard L. Sutton, Professor Emeritus of Dermatology, University of Kansas Medical School; and Richard L. Sutton, Jr., Assistant Professor of Dermatology, University of Kansas Medical School. Fourth edition. Pp. 904, with 723 illustrations. Cloth. Price $9.00. St. Louis: C. V. Mosby Company, 1941.

This text-book of dermatology represents an extensive revision of previous editions, with concise treatment of comparatively recent items, such as the "sulfamiracle" drugs and avitaminoses. The attached bibliography, including articles of 1940, brings the book thoroughly up to date.

The authors have adopted the plan of presenting salient features in large type; and including rare variations from the normal, and discussions, in smaller type, which may be read or omitted at the discretion of the reader. Illustrations, which are always of great value in a dermatological text-book, seem to be unusually clear and well selected for the feature to be depicted; and many of the pictures apparently are from the collection of the authors. The subject matter of the volume is handled in more or less orthodox fashion, beginning with introductory chapters on anatomy and physiology of the skin and general principles of treatment, to be followed by the simpler inflammations and purpuras, and ranging through the neuroses, pigmentations, neoplasms, to diseases caused by definite organisms.

The book is ambitious in its scope, and is to be recommended to those doing general practice, as well as to those engaged in the practice of dermatology.

Manual of Clinical Chemistry. By Miriam Reiner, M. Sc., Assistant Chemist, Mt. Sinai Hospital, New York. Pp. 296, with 18 illustrations. Price, $3.00. New York: Interscience Publishers, Inc., 1941.

The manual is intended for internes and laboratory technicians Any choice of analyt-

ical methods must remain the consideration of the hospital chemist, and equally important, of the doctor in charge of patients. The physiological and pathological interpretation of results of chemical analyses rests with the physician, who must, therefore, know the relative advantages and errors in any method he selects. The details of error and specificity of reactions are the field of the chemists. On these bases, the user of the manual, particularly if he be an interne, should read the two prefaces with care.

The choice of methods leans heavily to those based on colorimetric estimation of products of reaction between blood, urine, spinal fluid, feces and a variety of chemicals. I think that a paragraph or two on the specificity of colorimetric methods would have been desirable, in view of the intended audience; color is easy to see but not easy to correlate with certain chemical constituents of body fluids or body excreta. The rapidity of colorimetric estimations is a recommendation to most hospital authorities and to internes treating or merely studying patients in critical conditions; nevertheless, a rapid method of doubtful or untested specificity only seems to save time. The manual includes some methods which are open to this objection; I refer especially to the much used but unreliable colorimetric estimation of amino acids by the method of Folin. Recent modifications, particularly that of Sahuyun and Goodell, have improved the specificity, but even these better methods have so far failed to oust the older one, and they in turn are less quantitative than the nitrous acid manometric method of Van Slyke or the formal titration of Sorensen.

The manual would have been better had it separated methods used in clinical crises from those used mainly in the study of chronic diseases, and would have been very much better if errors, specificity, and interpretation of the "critical" methods had been set out in detail. To many internes and to most technicians a method is a method, something to be done without critical evaluation. Critical evaluation ought to be induced whenever the analysis bears on clinical crises.

With all these criticisms, the manual remains a useful handbook and contains many well tried methods. It is a start for the interne and a reference for the technician.

The Genuine Works of Hippocrates. Transcribed from the Greek by Francis Adams. Edited by Emerson Crosby Kelly, M. D. Pp. 384. Fabricoid. Price, $3.00. Baltimore: Williams & Wilkins Company, 1939.

Francis Adams, a Scottish surgeon, first published his translation in 1849; it immediately became a leading one, and now, nearly a century later, it still remains the most popular one. He faithfully preserves the Hippocratic style of writing—clear, precise, simple, with no semblance of self-laudation. After 2500 years, one is amazed at the powers of observation and description of the Father of Medicine.

Dr. Kelly's edition is confined to those writings regarded as genuine—a debatable theme even now. He has deleted most of Adams' lengthy technical footnotes, leaving a connected story that runs smoothly. The volume is a beautiful example of the bookmaker's art. Since Hippocrates is a "must" book on the list of every cultured physician, this edition should find wide acceptance.

The 1940 Year Book of General Surgery. Edited by Evarts A. Graham, M. D., Professor of Surgery, Washington University. Pp. 816, with 326 illustrations. Cloth. Price, $3.00. Chicago: Year Book Publishers, Inc., 1941.

This is the 40th Anniversary Edition of the Year Book on Surgery, and the 14th to be edited by Dr. Graham. The vast amount of work involved can be glimpsed by stating that it represents his selection of 767 articles from 107 publications in 11 countries! This is not a text-book; it is a summary and digest of the most significant surgical papers published in 1940, carefully selected and edited, with frequent critical annotations from the vast experience of the editor. Every region of the body is covered; 98 operative procedures (38 in detail) are included. There is a special section on Military Surgery. In a Special Foreword on Bronchiogenic Carcinoma Dr. Graham presents for the first time the results in 101 cases of his own.

The book is well printed, and the illustrations excellent. The index of authors is com-

plete, but the subject index could be enlarged with benefit.

If one should buy these Year Books each year he would have at his fingertips the best resume of modern surgical advances in the English language. This 1940 volume is unusually good.

How to Prevent Goiter. By Israel Bram. M. D., Medical Director, Bram Institute for Goiter and other Glandular Diseases. Pp. 182, with 21 illustrations. Cloth. Price, $2.00. New York: E. P. Dutton & Company, 1941.

Dr. Bram has been singularly successful in the medical treatment of goiter, and has written three books on the subject, for the profession. This volume, for the public, presents the subject of thyroid disease in simple language that any patient can understand and follow. The presentation and medical treatment of goiter is discussed in considerable detail, including diets, but the book makes no suggestion that the physician can be dispensed with. On the contrary, the author makes it plain that a competent M. D. must be on the job, and that this book is merely to assist the patient in understanding and following the doctor's orders better. As such an adjunct, the book is heartily recommended.

Macleod's Physiology in Modern Medicine. Edited by Philip Bard, M. D., Professor of Physiology, Johns Hopkins University School of Medicine. Ninth edition. Pp. 1256, with 387 illustrations. Cloth. Price, $10.00. St. Louis: C. V. Mosby Company, 1941.

It has been only three years since the last edition of this famous work appeared, yet such is the rapid advance of physiology that the editor and his nine collaborators found it necessary to rewrite many chapters. The general style of previous editions is maintained; secondary and controversial matters are in small type. The illustrations are good, the bibliography more than ample, and the index is excellent. The pale green paper, new in this edition, is welcomed. This "physiology" fully maintains the excellence and the completeness of its predecessors.